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NIXON & VANDERHYE, PC			KINSEY WHITE, NICOLE ERIN	
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Please find below and/or attached an Office communication concerning this application or proceeding.

The time period for reply, if any, is set in the attached communication.

Office Action Summary	Application No. 10/578,848	Applicant(s) HELLSTROM ET AL.
	Examiner NICOLE KINSEY WHITE	Art Unit 1648

-- The MAILING DATE of this communication appears on the cover sheet with the correspondence address --
Period for Reply

A SHORTENED STATUTORY PERIOD FOR REPLY IS SET TO EXPIRE 3 MONTH(S) OR THIRTY (30) DAYS, WHICHEVER IS LONGER, FROM THE MAILING DATE OF THIS COMMUNICATION.

- Extensions of time may be available under the provisions of 37 CFR 1.136(a). In no event, however, may a reply be timely filed after SIX (6) MONTHS from the mailing date of this communication.
- If no period for reply is specified above, the maximum statutory period will apply and will expire SIX (6) MONTHS from the mailing date of this communication.
- Failure to reply within the set or extended period for reply will, by statute, cause the application to become ABANDONED (35 U.S.C. § 133).

Any reply received by the Office later than three months after the mailing date of this communication, even if timely filed, may reduce any earned patent term adjustment. See 37 CFR 1.704(b).

Status

1) Responsive to communication(s) filed on **18 March 2010**.
 2a) This action is FINAL. 2b) This action is non-final.
 3) Since this application is in condition for allowance except for formal matters, prosecution as to the merits is closed in accordance with the practice under *Ex parte Quayle*, 1935 C.D. 11, 453 O.G. 213.

Disposition of Claims

4) Claim(s) **1,4-12,20 and 22** is/are pending in the application.
 4a) Of the above claim(s) _____ is/are withdrawn from consideration.
 5) Claim(s) _____ is/are allowed.
 6) Claim(s) **1,4-12,20 and 22** is/are rejected.
 7) Claim(s) _____ is/are objected to.
 8) Claim(s) _____ are subject to restriction and/or election requirement.

Application Papers

9) The specification is objected to by the Examiner.
 10) The drawing(s) filed on _____ is/are: a) accepted or b) objected to by the Examiner.
 Applicant may not request that any objection to the drawing(s) be held in abeyance. See 37 CFR 1.85(a).
 Replacement drawing sheet(s) including the correction is required if the drawing(s) is objected to. See 37 CFR 1.121(d).
 11) The oath or declaration is objected to by the Examiner. Note the attached Office Action or form PTO-152.

Priority under 35 U.S.C. § 119

12) Acknowledgment is made of a claim for foreign priority under 35 U.S.C. § 119(a)-(d) or (f).
 a) All b) Some * c) None of:
 1. Certified copies of the priority documents have been received.
 2. Certified copies of the priority documents have been received in Application No. _____.
 3. Copies of the certified copies of the priority documents have been received in this National Stage application from the International Bureau (PCT Rule 17.2(a)).

* See the attached detailed Office action for a list of the certified copies not received.

Attachment(s)

1) Notice of References Cited (PTO-892)
 2) Notice of Draftsperson's Patent Drawing Review (PTO-948)
 3) Information Disclosure Statement(s) (PTO/SB/08)
 Paper No(s)/Mail Date _____

4) Interview Summary (PTO-413)
 Paper No(s)/Mail Date _____

5) Notice of Informal Patent Application
 6) Other: _____

DETAILED ACTION

Claim Rejections - 35 USC § 103

The following is a quotation of 35 U.S.C. 103(a) which forms the basis for all obviousness rejections set forth in this Office action:

(a) A patent may not be obtained though the invention is not identically disclosed or described as set forth in section 102 of this title, if the differences between the subject matter sought to be patented and the prior art are such that the subject matter as a whole would have been obvious at the time the invention was made to a person having ordinary skill in the art to which said subject matter pertains. Patentability shall not be negated by the manner in which the invention was made.

This application currently names joint inventors. In considering patentability of the claims under 35 U.S.C. 103(a), the examiner presumes that the subject matter of the various claims was commonly owned at the time any inventions covered therein were made absent any evidence to the contrary. Applicant is advised of the obligation under 37 CFR 1.56 to point out the inventor and invention dates of each claim that was not commonly owned at the time a later invention was made in order for the examiner to consider the applicability of 35 U.S.C. 103(c) and potential 35 U.S.C. 102(e), (f) or (g) prior art under 35 U.S.C. 103(a).

Claims 1, 4-12, 20 and 22 are rejected under 35 U.S.C. 103(a) as being unpatentable over Neurath et al. (EP 154902A) and further in view of Zavaglia et al. (Italian Journal of Gastroenterology, 1996, 28(6):324-331, Abstract only) and Wei et al. (World J Gastroenterol, 2002;8(2):276-281).

The claims are directed to a method of determining whether an individual having hepatitis B virus (HBV) infection will respond to interferon alpha (IFN- α) treatment, the method comprising:

i) obtaining a pre-treatment sample from said HBV-infected individual, and
ii) analyzing said pre-treatment sample for the presence or absence of antibodies reactive with a preS 1 peptide consisting of the sequence of residues 94-117 (SEQ ID NO: 1) wherein the presence of said antibodies in said pre-treatment sample indicates that said individual will respond to said treatment and the absence of said antibodies in said pre-treatment sample indicates that said individual will not respond to said treatment.

The claims are further directed to treating chronically infected individuals, treating individuals who are HBeAg positive or negative and treating HBV infected individuals with corticosteroid.

Neurath et al. discloses a method for detecting the presence or absence of antibodies to pre-S of hepatitis B Virus in a sample, e.g., serum, comprising:

a) contacting the sample with a solid substrate coated with a non-labeled peptide containing an amino acid chain corresponding to at least six consecutive amino acids within the pre-S gene coded region of the envelope of HBV, the peptide free of an amino acid sequence corresponding to the naturally occurring envelope proteins of hepatitis B virus, incubating and washing said contacted sample;

b) contacting the incubated washed product obtained from step a above with a labeled peptide containing an amino acid chain corresponding to at least six consecutive amino acids within the pre-S gene coded region of the envelope of HBV, said peptide free of an amino acid sequence corresponding to the naturally occurring envelope protein of hepatitis B virus, incubating and washing the resultant mass; and

c) determining the extent of labeled peptide present in the resultant mass obtained by step b above (see page 15, line 14 to page 17, line 4).

Neurath et al. also teaches preferred peptides of the invention, including SEQ ID NO:1 (see page 31, lines 24-25 and claim 25) which can be used in the method of Neurath et al.

Neurath et al. also discloses a process for the detection of antibodies to proteins coded for by the pre-S region of hepatitis B virus DNA, comprising the following steps:

(a) adsorbing on a solid substrate containing binding sites thereon, e.g., polystyrene beads, a peptide having an amino acid sequence corresponding to at least six consecutive amino acids within the pre-S gene coded region of the HBV envelope,

(b) contacting the substrate from step a with a material to saturate the binding sites thereon,

c) washing the substrate from step b,

d) contacting the substrate from step c with a specimen comprising human sera,

(e) incubating the resultant mass of step d,

(f) washing the resultant mass of step e,

(g) adding radiolabeled antibodies to human IgG or IgM to the resultant mass of step f to form a second resultant mass,

(h) subjecting the second resultant mass of step g to counting in a gamma counter,

(i) subjecting normal sera utilized as a control to steps (a) to (h), and

(j) comparing the counts of steps h and i.

In the above process for the detection of antibodies, ELISA techniques can be substituted for RIA techniques (see page 20, line 14 to page 21, line 13).

Neurath et al. does not teach treating HBV infected individuals (HBeAg-HBV DNA-positive) with interferon- α alone or in combination with the corticosteroid, and Neurath et al. does not teach that the presence of preS1 antibodies indicates that the patient will respond to interferon alpha treatment.

Zavaglia et al. teaches treating HBV infected individuals (HBeAg-HBV DNA-positive) with interferon- α alone or in combination with the corticosteroid, deflazacort.

Zavaglia et al. found that serum HBV DNA levels decreased significantly in both groups.

Wei et al. teaches that the appearance of anti-preS1 antibody in the course of most acute hepatitis patients predicts the clearance of HBeAg and disappearance of preS1 dominants and HBV-DNA followed by elimination of HBsAg and seroconversion to anti-HBs. The role of anti-preS1 antibodies might be neutralization of HBsAg with preS1-coded epitopes (particularly infective HBV virions), as the antibodies were found in most cases of acute hepatitis followed by recovery. Anti-preS1 antibodies were hardly observed in patients with acute hepatitis progressing to chronic disease and in chronic hepatitis patients with continuing presence of preS1 domain and seropositive of HBeAg or anti-HBe. But anti-preS1 antibodies were detected in a few patients with chronic aggressive hepatitis undergoing treatment with antiviral agents, and the appearance of the antibodies correlated well with healthy improvement. The apparent prognostic implications of anti-preS1 antibodies are of interest in screening for this marker in hepatitis B patients. In conclusion, the presence of antibodies against preS1

in serum during acute infection may indicate subsequent recovery. Through detection of anti-preS1 antibodies based on biotin-labeled protein A indirect ELISA and follow-up study, it affords some information about the state and future prognosis of hepatitis B patients. The detection system has potential to be developed to a new kit for diagnosis and prognosis of hepatitis B patients (see page 280).

Wei et al. used the 21-119 region of preS1 because it contains several known epitopes of HBV (27-35aa, 72-78aa, 32-47aa, 41-53aa, 94-105aa, 106-117aa, 12-21aa, 21-30aa, 29-48aa and 94-117aa) (see page 276). This region contains applicants' epitope of interest.

Therefore, it would have been obvious to one of ordinary skill in the art to modify the methods taught by Neurath et al. and administer interferon- α alone or in combination with a corticosteroid to treat individuals where antibodies against preS1 were detected, whether the individual was HBeAg positive or negative. One would have been motivated to do so and there would have been a reasonable expectation of success given the fact that it is well known in the art to treat HBV with interferon- α and given the suggestion by Zavaglia et al. that interferon- α alone or in conjunction with a corticosteroid significantly decreases serum HBV DNA levels.

Further, it would have been obvious to one of ordinary skill in the art to combine the teachings of Wei et al. (i.e., the presence of antibodies against preS1 during acute infection and during chronic aggressive infection in patients undergoing treatment may indicate subsequent recovery and correlates with a healthy improvement) with the method of Neurath et al. to predict or determine a positive response (or recovery) to

HBV treatment with antiviral agents (e.g., interferon), especially since the patient is already on his/her way to recovery based on the presence of anti-preS1 antibodies. One would have been motivated to do so and there would be a reasonable expectation of success given the findings of Wei et al.

Thus, the invention as a whole was clearly *prima facie* obvious to one of ordinary skill in the art at the time the invention was made.

Claims 1, 4-12, 20 and 22 are rejected under 35 U.S.C. 103(a) as being unpatentable over Neurath et al. (EP 448126A) and further in view of Zavaglia et al. (Italian Journal of Gastroenterology, 1996, 28(6):324-331, Abstract only) and Wei et al. (World J Gastroenterol, 2002;8(2):276-281).

Neurath et al. discloses a method for detecting the presence or absence of antibodies to pre-S of hepatitis B Virus in a test sample, e.g., serum (see page 8, lines 6-50 and page 9, lines 9-53).

Neurath et al. also discloses preferred peptides of the invention, including instant SEQ ID NO:1 (see page 13, lines 30-31) that can be used in the method.

The method of Neurath et al. for the detection of antibodies to proteins coded for by the pre-S region of hepatitis B virus DNA, comprising the following steps:

(a) adsorbing on a solid substrate containing binding sites thereon, e.g., polystyrene beads, a peptide having an amino acid sequence corresponding to at least six consecutive amino acids within the pre-S gene coded region of the HBV envelope,

(b) contacting the substrate from step a with a material to saturate the binding sites thereon,

c) washing the substrate from step b,

d) contacting the substrate from step c with a specimen comprising human sera,

(e) incubating the resultant mass of step d,

(f) washing the resultant mass of step e,

(g) adding radiolabeled antibodies to human IgG or IgM to the resultant mass of step f to form a second resultant mass,

(h) subjecting the second resultant mass of step g to counting in a gamma counter,

(i) subjecting normal sera utilized as a control to steps (a) to (h), and

(j) comparing the counts of steps h and i.

Neurath et al. does not teach treating HBV infected individuals (HBeAg-HBV DNA-positive) with interferon- α alone or in combination with the corticosteroid, and Neurath et al. does not teach that the presence of preS1 antibodies indicates that the patient will respond to interferon alpha treatment.

Zavaglia et al. teaches treating HBV infected individuals (HBeAg-HBV DNA-positive) with interferon- α alone or in combination with the corticosteroid, deflazacort. Zavaglia et al. found that serum HBV DNA levels decreased significantly in both groups.

Wei et al. teaches that the appearance of anti-preS1 antibody in the course of most acute hepatitis patients predicts the clearance of HBeAg and disappearance of preS1 dominants and HBV-DNA followed by elimination of HBsAg and seroconversion

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to anti-HBs. The role of anti-preS1 antibodies might be neutralization of HBsAg with preS1-coded epitopes (particularly infective HBV virions), as the antibodies were found in most cases of acute hepatitis followed by recovery. Anti-preS1 antibodies were hardly observed in patients with acute hepatitis progressing to chronic disease and in chronic hepatitis patients with continuing presence of preS1 domain and seropositive of HBeAg or anti-HBe. But anti-preS1 antibodies were detected in a few patients with chronic aggressive hepatitis undergoing treatment with antiviral agents, and the appearance of the antibodies correlated well with healthy improvement. The apparent prognostic implications of anti-preS1 antibodies are of interest in screening for this marker in hepatitis B patients. In conclusion, the presence of antibodies against preS1 in serum during acute infection may indicate subsequent recovery. Through detection of anti-preS1 antibodies based on biotin-labeled protein A indirect ELISA and follow-up study, it affords some information about the state and future prognosis of hepatitis B patients. The detection system has potential to be developed to a new kit for diagnosis and prognosis of hepatitis B patients (see page 280).

Wei et al. used the 21-119 region of preS1 because it contains several known epitopes of HBV (27-35aa, 72-78aa, 32-47aa, 41-53aa, 94-105aa, 106-117aa, 12-21aa, 21-30aa, 29-48aa and 94-117aa) (see page 276). This region contains applicants' epitope of interest.

Therefore, it would have been obvious to one of ordinary skill in the art to modify the methods taught by Neurath et al. and administer interferon- α alone or in combination with a corticosteroid to treat individuals where antibodies against preS1

were detected, whether the individual was HBeAg positive or negative. One would have been motivated to do so and there would have been a reasonable expectation of success given the fact that it is well known in the art to treat HBV with interferon- α and given the suggestion by Zavaglia et al. that interferon- α alone or in conjunction with a corticosteroid significantly decreases serum HBV DNA levels.

Further, it would have been obvious to one of ordinary skill in the art to combine the teachings of Wei et al. (i.e., the presence of antibodies against preS1 during acute infection and during chronic aggressive infection in patients undergoing treatment may indicate subsequent recovery and correlates with a healthy improvement) with the method of Neurath et al. to predict or determine a positive response (or recovery) to HBV treatment with antiviral agents (e.g., interferon), especially since the patient is already on his/her way to recovery based on the presence of anti-preS1 antibodies. One would have been motivated to do so and there would be a reasonable expectation of success given the findings of Wei et al.

Thus, the invention as a whole was clearly *prima facie* obvious to one of ordinary skill in the art at the time the invention was made.

Claims 1, 4-12, 20 and 22 are rejected under 35 U.S.C. 103(a) as being unpatentable over Neurath et al. (U.S. Patent No. 4,847,080) and further in view of Zavaglia et al. (Italian Journal of Gastroenterology, 1996, 28(6):324-331, Abstract only) and Wei et al. (World J Gastroenterol, 2002;8(2):276-281).

Neurath et al. discloses a method for the detection of antibodies to proteins coded for by the pre-S region of hepatitis B virus DNA. The method comprises:

- (a) adsorbing on a solid substrate containing binding sites thereon, e.g., polystyrene beads, a peptide having an amino acid sequence corresponding to at least six consecutive amino acids within the pre-S gene coded region of the HBV envelope,
- (b) contacting the substrate from step a with a material to saturate the binding sites thereon,
- c) washing the substrate from step b,
- d) contacting the substrate from step c with a specimen comprising human sera,
- (e) incubating the resultant mass of step d,
- (f) washing the resultant mass of step e,
- (g) adding radiolabeled antibodies to human IgG or IgM to the resultant mass of step f to form a second resultant mass,
- (h) subjecting the second resultant mass of step g to counting in a gamma counter,
- (i) subjecting normal sera utilized as a control to steps (a) to (h), and
- (j) comparing the counts of steps h and i.

In the above process for the detection of antibodies, ELISA techniques can be substituted for RIA techniques (col. 9, lines 3-31).

Neurath et al. also teaches preferred peptides of the invention, including SEQ ID NO:1 (col. 14, lines 8-10 and claim 22) that can be used in the method.

Neurath et al. does not teach treating HBV infected individuals (HBeAg-HBV DNA-positive) with interferon- α alone or in combination with the corticosteroid, and Neurath et al. does not teach that the presence of preS1 antibodies indicates that the patient will respond to interferon alpha treatment.

Zavaglia et al. teaches treating HBV infected individuals (HBeAg-HBV DNA-positive) with interferon- α alone or in combination with the corticosteroid, deflazacort. Zavaglia et al. found that serum HBV DNA levels decreased significantly in both groups.

Wei et al. teaches that the appearance of anti-preS1 antibody in the course of most acute hepatitis patients predicts the clearance of HBeAg and disappearance of preS1 dominants and HBV-DNA followed by elimination of HBsAg and seroconversion to anti-HBs. The role of anti-preS1 antibodies might be neutralization of HBsAg with preS1-coded epitopes (particularly infective HBV virions), as the antibodies were found in most cases of acute hepatitis followed by recovery. Anti-preS1 antibodies were hardly observed in patients with acute hepatitis progressing to chronic disease and in chronic hepatitis patients with continuing presence of preS1 domain and seropositive of HBeAg or anti-HBe. But anti-preS1 antibodies were detected in a few patients with chronic aggressive hepatitis undergoing treatment with antiviral agents, and the appearance of the antibodies correlated well with healthy improvement. The apparent prognostic implications of anti-preS1 antibodies are of interest in screening for this marker in hepatitis B patients. In conclusion, the presence of antibodies against preS1 in serum during acute infection may indicate subsequent recovery. Through detection of anti-preS1 antibodies based on biotin-labeled protein A indirect ELISA and follow-up

study, it affords some information about the state and future prognosis of hepatitis B patients. The detection system has potential to be developed to a new kit for diagnosis and prognosis of hepatitis B patients (see page 280).

Wei et al. used the 21-119 region of preS1 because it contains several known epitopes of HBV (27-35aa, 72-78aa, 32-47aa, 41-53aa, 94-105aa, 106-117aa, 12-21aa, 21-30aa, 29-48aa and 94-117aa) (see page 276). This region contains applicants' epitope of interest.

Therefore, it would have been obvious to one of ordinary skill in the art to modify the methods taught by Neurath et al. and administer interferon- α alone or in combination with a corticosteroid to treat individuals where antibodies against preS1 were detected, whether the individual was HBeAg positive or negative. One would have been motivated to do so and there would have been a reasonable expectation of success given the fact that it is well known in the art to treat HBV with interferon- α and given the suggestion by Zavaglia et al. that interferon- α alone or in conjunction with a corticosteroid significantly decreases serum HBV DNA levels.

Further, it would have been obvious to one of ordinary skill in the art to combine the teachings of Wei et al. (i.e., the presence of antibodies against preS1 during acute infection and during chronic aggressive infection in patients undergoing treatment may indicate subsequent recovery and correlates with a healthy improvement) with the method of Neurath et al. to predict or determine a positive response (or recovery) to HBV treatment with antiviral agents (e.g., interferon), especially since the patient is already on his/her way to recovery based on the presence of anti-preS1 antibodies.

One would have been motivated to do so and there would be a reasonable expectation of success given the findings of Wei et al.

Thus, the invention as a whole was clearly *prima facie* obvious to one of ordinary skill in the art at the time the invention was made.

Response to Arguments

In the reply dated March 18, 2010, applicants make several arguments which are addressed below.

First, applicants argue that the combination of Neurath et al., Zavaglia et al. and Wei et al. does not provide a teaching or suggestion that antibodies to 94-117 (SEQ ID NO:1) would predict a response to IFN therapy. Applicants' arguments have been fully considered but not found persuasive.

As outlined above, Wei et al. teaches that B-cell epitopes of preS1 have been mapped to residues 27-35aa, 72-78aa, 32-47aa, 41-53aa, 94-105aa, 106-117aa, and T-cell epitopes have been mapped to 12-21aa, 21-30aa, 29-48aa and 94-117aa (instant SEQ ID NO:1). These epitopes have been extensively analyzed and fall within residues 21-119aa.

Wei et al. used the 21-119aa fragment in immunoassays to detect anti-preS1 antibodies. Results showed that more than half of 19 acute hepatitis B patients produced anti-preS1 antibodies during recovery of the disease. Wei et al. also found that in subjects undergoing treatment, the appearance of anti-preS1 antibodies correlated well with improvement in health. Wei et al. states that anti-preS1 antibodies

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were detected in a few patients with chronic aggressive hepatitis undergoing treatment with antiviral agents, and the appearance of the antibodies correlated well with healthy improvement (see page 280). Wei et al. goes on to state that the apparent prognostic implications of anti-preS1 antibodies are of interest in screening for this marker in hepatitis B patients. In conclusion, the study suggested that presence of antibodies against preS1(21-119aa) region in serum during acute infection may indicate subsequent recovery. Through detection of anti-preS1(21- 19aa) antibodies, it afforded some information about the state and future prognosis of hepatitis B patients. The detection system has potential to be developed to a new kit for diagnosis and prognosis of hepatitis B patients (see page 280).

Neurath et al. discloses a method for detecting in a sample the presence or absence of antibodies to pre-S fragments of hepatitis B Virus, including instant SEQ ID NO:1.

Zavaglia et al. teaches treating HBV infected individuals (HBeAg-HBV DNA-positive) with interferon- α alone or in combination with the corticosteroid, deflazacort. Zavaglia et al. found that serum HBV DNA levels decreased significantly in both groups.

As outlined in the rejections above, it would have been obvious to one of ordinary skill in the art to combine the teachings of Wei et al. (i.e., the presence of antibodies against preS1 known epitopes during acute infection and during chronic aggressive infection in patients undergoing treatment may indicate subsequent recovery and correlates with a healthy improvement) with the antibody detection methods of Neurath et al. to detect preS1 antibodies and predict or determine a positive outcome (or

recovery) for HBV treatment with antiviral agents known in the art (e.g., interferon, lavumidine, etc.), especially since the patient is already on his/her way to recovery based on the presence of anti-preS1 antibodies. One would have been motivated to do so and there would be a reasonable expectation of success given the findings of Wei et al.

Applicants' arguments that Wei et al. detected the whole population of antibodies to all of the epitopes present in the 21-119 fragment and that Wei et al. is silent about antibodies to preS1 fragment 94-117 have been fully considered but not found persuasive. Wei et al. discloses fragment 94-117 (instant SEQ ID NO:1) as an epitope within the 21-119 preS1 fragment that produced antibodies that are predictive of a positive outcome. Although Wei et al. detected the whole population of antibodies to the epitopes present in the 21-119 preS1 fragment, Wei et al. provides one of ordinary skill in the art with a list of each epitope within the 21-119 fragment (including instant SEQ ID NO:1) and the location of each epitope so that the skilled artisan, if he or she desires, can easily map the antibodies in the population to the epitopes of 21-119 and determine which epitopes were represented in the population of antibodies. Applicants have merely taken what Wei et al. discloses and gone one step further by characterizing the antibody population by mapping the antibodies to the 94-117 epitope already disclosed by Wei et al.

No claim is allowed.

THIS ACTION IS MADE FINAL. Applicant is reminded of the extension of time policy as set forth in 37 CFR 1.136(a).

A shortened statutory period for reply to this final action is set to expire THREE MONTHS from the mailing date of this action. In the event a first reply is filed within TWO MONTHS of the mailing date of this final action and the advisory action is not mailed until after the end of the THREE-MONTH shortened statutory period, then the shortened statutory period will expire on the date the advisory action is mailed, and any extension fee pursuant to 37 CFR 1.136(a) will be calculated from the mailing date of the advisory action. In no event, however, will the statutory period for reply expire later than SIX MONTHS from the mailing date of this final action.

Any inquiry concerning this communication or earlier communications from the examiner should be directed to NICOLE KINSEY WHITE whose telephone number is (571)272-9943. The examiner can normally be reached on Monday through Friday from 9:00 am to 5:30 pm.

If attempts to reach the examiner by telephone are unsuccessful, the examiner's supervisor, Jeffrey Stucker can be reached on (571) 272-0911. The fax phone number for the organization where this application or proceeding is assigned is 571-273-8300.

Information regarding the status of an application may be obtained from the Patent Application Information Retrieval (PAIR) system. Status information for published applications may be obtained from either Private PAIR or Public PAIR. Status information for unpublished applications is available through Private PAIR only. For more information about the PAIR system, see <http://pair-direct.uspto.gov>. Should you have questions on access to the Private PAIR system, contact the Electronic Business Center (EBC) at 866-217-9197 (toll-free). If you would like assistance from a USPTO Customer Service Representative or access to the automated information system, call 800-786-9199 (IN USA OR CANADA) or 571-272-1000.

/Nicole Kinsey White/
Examiner, Art Unit 1648

/Stacy B. Chen/
Primary Examiner, Art Unit 1648